

Facial Nerve

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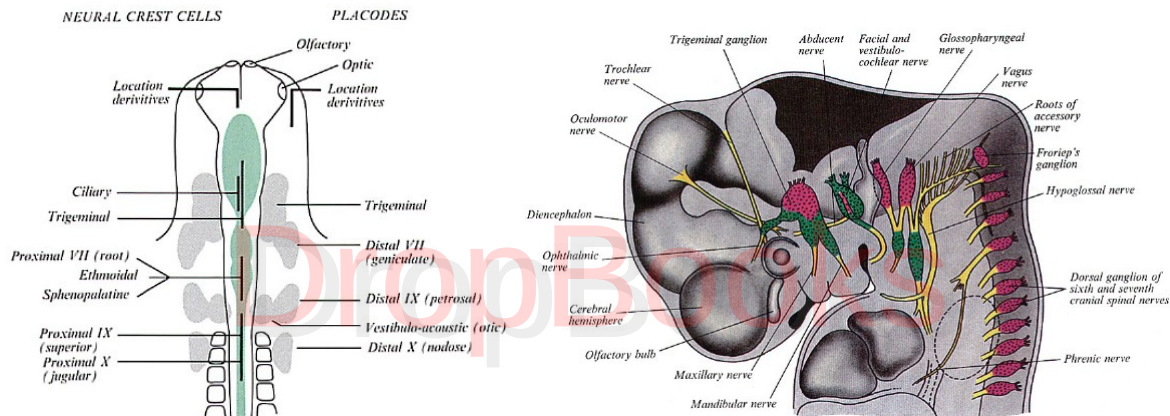
Embryology:

Embryology of the facial nerve is complex.

DEVELOPMENTAL ANATOMY

Weeks 0-4

- The rhombencephalon is divided into the myelencephalon (caudal), which becomes the medulla oblongata, and the metencephalon (cranial).
- The facial nerve lies between the myelencephalon and metencephalon.
- By the end of the 4th week, the facial nerve splits into 2 parts.
- The chorda tympani nerve exits courses ventrally to the 1st pharyngeal pouch to enter the mandibular arch. The motor fibers of the facial nerve migrate near the floor of the developing 4th ventricle. The fascio-acoustic primordium appears during the 3rd week.



Weeks 5-6

- Mesenchymal concentrations (form the cephalic muscles) are seen in association with their nerves, and the geniculate ganglion is identifiable.
- The greater superficial petrosal nerve (GSPN) is present. It courses to the lateral aspect of the developing internal carotid artery (ICA), where it joins the deep petrosal nerve and continues as the nerve of the pterygoid canal. It terminates in a group of cells that will become the pterygopalatine ganglion.
- The chorda tympani nerve in the mandibular arch terminates near a branch of the trigeminal nerve that will become the lingual nerve. The posterior auricular nerve appears near the chorda tympani.
- Complete separation of the facial and acoustic nerves. A discrete nervus intermedius develops.

Week 7

- The parotid bud is appearing unbranched, as an evagination from the lateral oral cavity.
- By the time, first order ductules of the parotid primordium are present. Gasser elegantly depicted the intricate nature of the development of the parotid gland and the facial nerve.
- All the peripheral branches can be identified, but the temporal branches have not yet reached the frontal region.

- The following facial muscles appear: *zygomaticus major, depressor anguli oris, buccinator, frontalis, and zygomaticus minor*.

Week 8

- A sulcus develops around the facial nerve; this is the beginning of the *fallopian canal*.
- *Orbicularis oris, levator anguli oris, and orbicularis oculi* muscles appear.

Week 9

- *Auricularis anterior, corrugator supercilii, occipital and mandibular platysma, and levator labii superioris* muscles appear.

Weeks 10-15

- Extensive branching of the peripheral portions of the facial nerve occurs.
- Complicated anatomy of the superficial and deep lobes of the parotid and their relation to the facial nerve are evident.

Week 16 to birth

- All definitive communications of the facial nerve are established by the 16th week.
- In late fetal life, the fallopian canal is closed by bone in most areas, except in the *anterior cranial portion*, where it remains open to form the *facial hiatus* along the floor of the middle cranial fossa. At least 25% and as many as 55% of fallopian canals have dehiscences; the most common site is adjacent to the oval window.
- At birth, the anatomy of the facial nerve approximates that of the adult, except for its exit through the more superficially located stylomastoid foramen. Adult anatomy will occur in this region as the mastoid tip develops after birth.

Summary

- By the 3rd week of gestation, the fascioacoustic primordium gives rise to cranial nerves VII and VIII.
- During the 4th week, the chorda tympani can be discerned from the main branch. The former courses ventrally into the first branchial arch and terminates near a branch of the trigeminal nerve that eventually becomes the lingual nerve.
- The main trunk courses into the mesenchyme, approaching the epibranchial placode. The geniculate ganglion, nervus intermedius, and the greater superficial petrosal nerve are visible by the 5th week.
- The second branchial arch gives rise to the muscles of facial expression in the seventh and 8th week.
- To innervate these muscles, the facial nerve courses across the region that eventually becomes the middle ear.
- By the 11th week, the facial nerve has arborized extensively. In the newborn, the facial nerve anatomy approximates that of an adult, except for its location in the mastoid, which is more superficial.

CONGENITAL FACIAL PARALYSIS

- Abnormalities of the facial nerve may occur in conjunction with *malformations of the ear*, in isolation without associated anomalies, or in conjunction with a variety of syndromes that include abnormalities elsewhere in the body.
- 1 in 2000 has a unilateral facial palsy, with a 90% spontaneous recovery rate.
- 75-80% are related to birth trauma (forceps delivery, prolonged labor, or ecchymosis over the mastoid or hemotympanum).
- Bilateral facial paralysis, other cranial nerve deficits, or other anomalies suggests a developmental etiology.
- Evaluation includes EEMG, CT scan, and EMG.

- If the etiology is traumatic, the nerve can be stimulated for 3-5 days postnatal; fibrillation potentials on EMG develop 14-21 days after birth.
- If the cause is not traumatic, treatment generally is delayed. Eye protection rarely is required in congenital facial paralysis.
- Many hereditary conditions include the deformity of facial paralysis. In addition, many hereditary and congenital malformations are associated with abnormal facial nerve anatomy in the presence of normal nerve function. (e.g. Möbius Syndrome, Goldenhar syndrome, dystrophia myotonica, and Mielke syndrome).

Developmental Syndromes Associated With Facial Nerve Abnormalities

Syndrome	Facial Nerve Abnormality	Description
DiGeorge syndrome	Facial paralysis reported	Multiple anomalies of craniofacial, cardiovascular, and visceral structures; absent and/or hypoplastic thymus and parathyroid glands Abnormal aural development
Dominant craniometaphyseal dysplasia	Unilateral and bilateral facial paralysis reported	AD Metaphyseal widening of limbs and bony overgrowth of facial bones and skull Obliteration of mastoid air cells Conductive and sensorineural hearing loss Manifest in early infancy or childhood
Recessive craniometaphyseal dysplasia	Unilateral facial paralysis	AR bone disease Major features - Glabella and paranasal prominence with severe mandibular prognathism Nasal obstruction Ocular hypertelorism Severe hearing loss
Dystrophia myotonica	Lack of facial animation	Progressive familial distal myopathy Affects muscles of jaw, face, neck, and eyelids
Hemifacial microsomia	Facial asymmetry	Unilateral microtia, macrostomia, and failure of mandibular ramus and condyle to form
Hereditary acoustic neuromas	Facial paresis and/or palsy	AD Manifests in third decade Symptoms 2 nd to tumor encroachment on nerves
Möbius syndrome	Bilateral facial paralysis Masklike facies	Unilateral or bilateral abducens paralysis + Poland syndrome
Sickle cell disease	Facial paralysis observed to occur during a crisis	Hemoglobinopathy Recurrent attacks of fatigue, weakness, abdominal pain, anorexia, and jaundice May have sensorineural hearing loss during a crisis
von Recklinghausen neurofibromatosis	Facial paralysis possible from a neurofibroma of the facial nerve or secondary to encroachment by an acoustic schwannoma	AD Multiple skin tumors, cutaneous pigmentation (cafe-au-lait spots), Incidence of 1 in 2000 Malignant degeneration in 3-12%

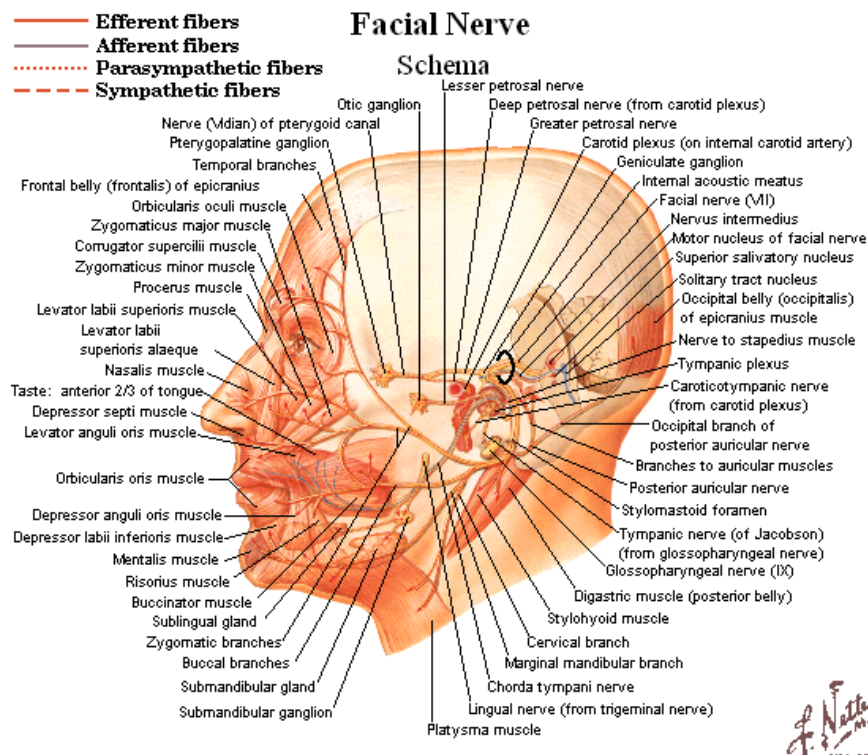
Summary of the Derivative of the 2nd Branchial Arch

Pharyngeal Arch	Nerve	Artery	Muscles	Skeleton
II = Hyoid (Reichert cartilage)	Cranial nerve (CN) VII = Facial nerve	Stapedial	Muscles of facial expression Buccinator Posterior belly of digastric Stylohyoid muscle Stapedius muscle	Manubrium of malleus Long process incus Stapes (except for footplate) Facial canal Styloid process Stylohyoid ligament Lesser cornu of

Facial Nerve Anatomy

OVERVIEW

- It is the nerve of facial expression. The pathways of the facial nerve are variable, and knowledge of the key intratemporal and extratemporal landmarks is essential for accurate physical diagnosis and safe and effective surgical intervention in the head and neck.



- It is composed of approximately 10,000 neurons:
 - 7,000 of which are myelinated and innervate the nerves of facial expression.
 - 3,000 of the nerve fibers are somatosensory and secretomotor and comprise the nervus intermedius.
- The course of the facial nerve and its central connections can be roughly divided into 6 segments:

Segment	Location	Length, mm
Supranuclear	Cerebral cortex	NA
Brain stem	Motor nucleus of facial nerve, superior salivatory nucleus of tractus solitarius	NA
Meatal segment	Brain stem to IAC	13-15
Labyrinthine segment	Fundus of IAC to facial hiatus	3-4
Tympanic segment	Geniculate ganglion to pyramidal eminence	8-11
Mastoid segment	Pyramidal process to stylomastoid foramen	10-14
Extratemporal segment	Stylomastoid foramen to pes anserinus	15-20

CENTRAL CONNECTIONS

- The voluntary responses of the facial muscles (eg, smiling when taking a photograph) arise from efferent discharge from the motor face area of the cerebral cortex → fascicles of the corticobulbar tract → the internal capsule → upper midbrain → lower brain stem where they synapse in the pontine facial nerve nucleus → divided into an upper and a lower half, bilaterally.
- A cortical lesion that produces a lower facial deficit is usually associated with a motor deficit of the tongue and weakness of the thumb, fingers, or hand on the ipsilateral side.
- Nerve fibers influencing emotional facial expression are thought to arise in the thalamus and globus pallidus. Supranuclear pyramidal lesions spare movements of the face initiated as emotional responses and reflexes. With nuclear and infranuclear lesions, loss of both involuntary and voluntary facial movement occurs.
- The facial nerve nuclei also receive afferent input from other brainstem nuclei. Input from the trigeminal nerve and nucleus form the basis of the trigeminofacial reflexes, eg, the corneal reflex. Input from the acoustic nuclei to the facial nerve nucleus forms part of the stapedial reflex response to loud noises.

Syndromes Associated with Central Lesions

Syndrome	Location of Lesion	Characteristic Feature
Foville syndrome	Lateral pons	Ipsilateral facial paresis, ipsilateral facial analgesia, ipsilateral Homer syndrome, ipsilateral deafness
Meige syndrome	Basal ganglion	Facial dystonia
Millard-Gubler syndrome	Pontine nucleus	Unilateral sixth nerve palsy, ipsilateral seventh nerve palsy, contralateral hemiparesis
Möbius syndrome	Fundus of IAC to facial hiatus	Ipsilateral facial paresis, ipsilateral abducens (CN VI) palsy
Parkinson disease	Extrapyramidal pathways	Masked facies
Pseudobulbar palsy	Pontine	Bilateral facial paresis with other CN defects, hyperactive gag reflex, hyperreflexia associated with hypertension, emotional lability
Weber syndrome	Upper midbrain	Ipsilateral loss of direct and consensual pupillary light reflexes, ipsilateral external strabismus, oculomotor paresis

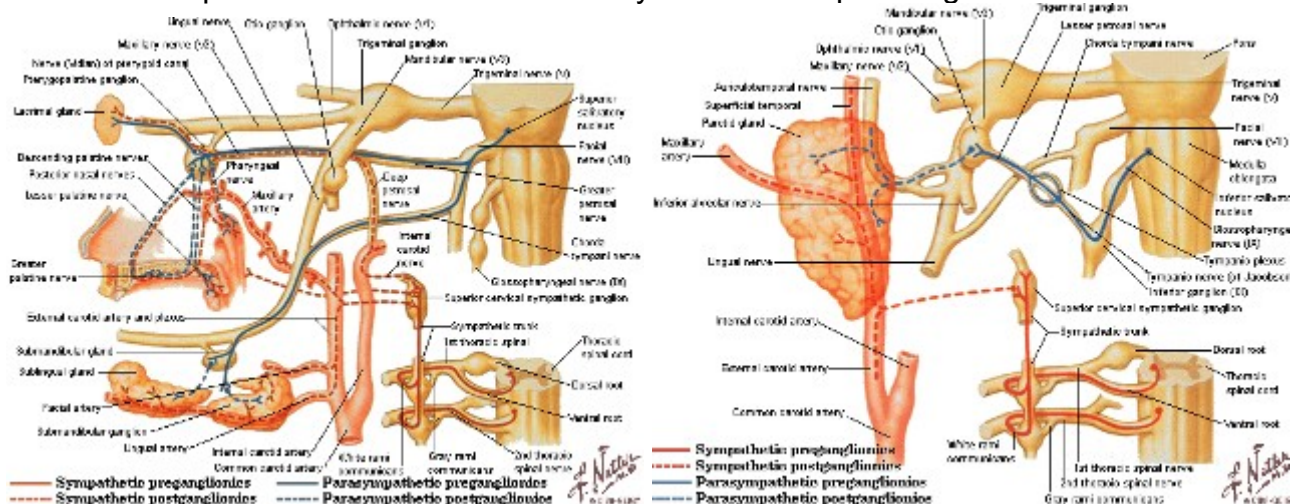
CEREBELLOPONTINE ANGLE AND THE INTERNAL AUDITORY CANAL

- The facial nerve emerges from the brain stem with the nerve of Wrisberg, ie, the nervus intermedius
- The nervus intermedius conveys:
 - (1) afferent taste fibers from the chorda tympani nerve, (anterior $\frac{2}{3}$ of the tongue);
 - (2) afferent taste fibers from the soft palate via the palatine and greater petrosal nerves;
 - (3) preganglionic parasympathetic innervation to the submandibular, sublingual, and lacrimal glands.
 - (4) afferent small cutaneous sensory component from the auricle and postauricular area.
- The fibers for taste originate in the nucleus of the tractus solitarius (NTS), and the fibers to the lacrimal, nasal, palatal mucus, and submandibular glands originate in the superior salivatory nucleus. Fibers to the lacrimal gland are carried with the greater superficial petrosal nerve until it exits the skull, where they branch off as the Vidian nerve (nerve of pterygoid canal) to be carried by V2 of trigeminal nerve.

- The close anatomic association between the facial nerve, the nervus intermedius, and the vestibulocochlear nerve at the level of the CPA and in the IAC may result in disturbances in tearing, taste, salivary gland flow, hearing, balance, and facial function as the result of lesions at this level.

INTRATEMPORAL COURSE OF THE FACIAL NERVE

- The facial nerve travels through the petrous temporal bone in the fallopian canal. Because of this bony shell around the nerve, inflammatory processes involving the CNS, facial nerve, and traumatic injuries to the temporal bone can produce unique complications.
- The labyrinthine segment is the narrowest part of the facial nerve and is susceptible to compression by means of edema. This is the only segment of the facial nerve that lacks anastomosing arterial cascades, making the area vulnerable to embolic phenomena, low-flow states, or vascular compression.
- After traversing the labyrinthine segment, the facial nerve changes direction to form the first genu, marking the location of the geniculate ganglion. Three nerves branch from the geniculate ganglion: the greater superficial petrosal nerve, the lesser petrosal nerve, and the external petrosal nerve.
- The GSPN emerges from the upper portion of the ganglion and carries secretomotor fibers to the lacrimal gland. (petrous temporal bone→greater petrosal foramen→middle cranial fossa→deep to trigeminal ganglion→foramen lacerum→pterygoid canal→ joins the deep petrosal nerve→nerve of the pterygoid canal→Axons from this nerve synapse in the pterygopalatine ganglion→ postganglionic parasympathetic fibers→are carried via branches of the maxillary (V2) divisions of the trigeminal nerve→lacrimal gland and mucus glands of the nasal and oral cavities.
- The lesser petrosal nerve carries secretory fibers to the parotid gland.



➤ Tympanic or horizontal segment

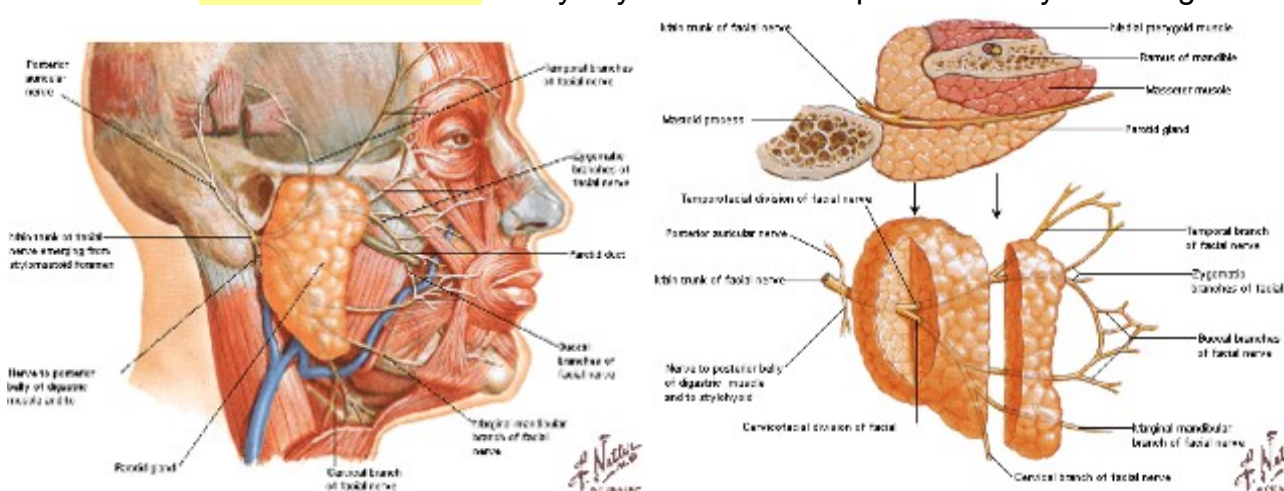
- The tympanic segment extends from the geniculate ganglion to the horizontal semicircular canal and is 8-11 mm in length. The nerve passes behind the cochleariform process and the tensor tympani
- The distal portion of the facial nerve emerges from the middle ear between the posterior wall of the external auditory canal and the horizontal semicircular canal. This is just distal to the pyramidal eminence, where the facial nerve makes a second genu. The most important landmarks for identifying the facial nerve in the mastoid are the horizontal semicircular canal, the fossa incudis, and the digastric ridge.
- The second genu of the facial nerve runs inferolateral to the lateral semicircular canal. This is a relatively constant relationship.

- The digastric ridge points to the lateral and inferior aspect of the vertical course of the facial nerve in the temporal bone. In poorly pneumatized temporal bones, the digastric ridge may be difficult to identify. The chorda tympani nerve and the fossa incudis can be used to identify the nerve when performing a facial recess approach
- **Mastoid segment**
- From second genu nerve continues vertically down the anterior wall of the mastoid process to the stylomastoid foramen.
- The mastoid segment is the longest part of the intratemporal course of the facial nerve, approximately 10-14 mm long. During middle ear surgery, the facial nerve is most commonly injured at the pyramidal turn.
- The 3 branches that exit from the mastoid segment of the facial nerve are:
 - (1) the nerve to the stapedius muscle,
 - (2) the chorda tympani nerve,
 - (3) the nerve from the auricular branch of the vagus. (arises from the jugular foramen and joins the facial nerve just distal to the point at which the nerve to the stapedius muscle arises. Pain fibers to the posterior auditory canal may be carried with this nerve).
- The chorda tympani is the terminal branch of the nervus intermedius. The chorda runs laterally in the middle ear, between the incus and malleus (crosses the middle ear cavity) and exits through the petrotympanic fissure (ie, canal of Huguier) to join the lingual nerve.
- The chorda tympani nerve carries:
 1. preganglionic secretomotor fibers to the submaxillary and sublingual glands.
 2. special sensory afferent fibers (ie, taste fibers) from the anterior $\frac{2}{3}$ of tongue
 3. fibers from the posterior wall of the external auditory canal responsible for pain, temperature, and touch sensations.
- The facial nerve exits the fallopian canal via the stylomastoid foramen. The nerve travels between the digastric and stylohyoid muscles and enters the parotid gland. A sensory branch exits the nerve just below the stylomastoid foramen and innervates the posterior wall of the external auditory canal and a portion of the tympanic membrane.

EXTRATEMPORAL FACIAL NERVE

- A number of useful landmarks are used to locate the facial nerve.
- Topographic landmarks:
 - a line drawn between the mastoid tip and the angle of the mandible can serve as a useful landmark for the superior limits of a neck dissection. (Removal of parotid tissue inferior to this line can be performed relatively safely).
 - The topographic trajectory of the frontal and/or marginal branches should be identified during a rhytidoplasty, submandibular gland excision, and/or neck dissection.
 - The frontal branch can be roughly located along a line extending from the attachment of the lobule (approximately 5 mm below the tragus), anterior and superiorly to a point 1.5 cm above the lateral aspect of the ipsilateral eyebrow.
- Surgical landmarks:
 - the tympanomastoid suture line: The main trunk (lies between the mastoid and tympanic segments of the temporal bone, 6-8 mm lateral to the stylomastoid foramen).

- the tragal pointer The main trunk (midway between (10 mm posteroinferior) the cartilaginous tragal pointer of the EAC and the posterior belly of the digastric muscle).
 - the posterior belly of the digastric muscle.
 - the surgeon may encounter a branch from the occipital artery that lies lateral to the nerve. Brisk bleeding at this time may be a sign that the nerve is in close proximity; hemostasis should be obtained using bipolar electrocautery, and further dissection should proceed cautiously.
 - The styloid process is deep to the main trunk of the nerve.
 - In the infant and young child, these landmarks are not applicable (facial nerve is located more superficially),
- Once it has exited stylomastoid foramen, the facial nerve gives off several rami before it divides into its main branches.
- The **posterior auricular nerve** → the postauricular muscles & occipitalis.
 - **Two small branches** → stylohyoid muscle and posterior belly of the digastric.



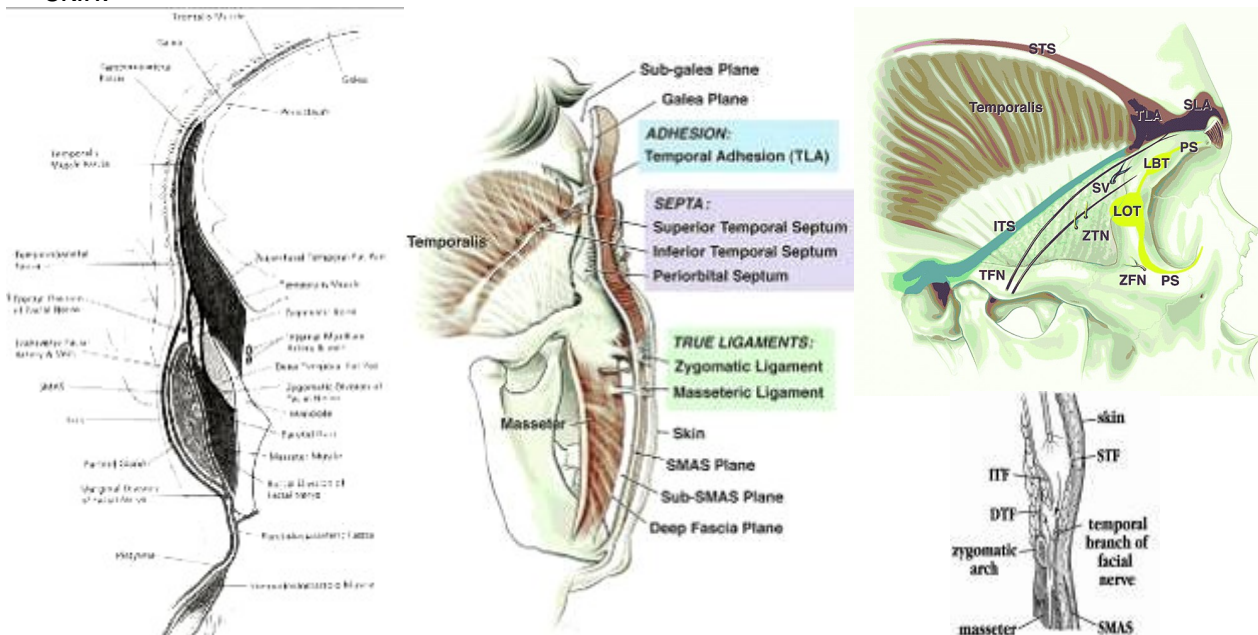
- The facial nerve crosses lateral to the styloid process and penetrates the parotid gland. The nerve lies in a **fibrous plane** that separates the deep and superficial lobes of the parotid gland → divides at the *pes anserinus* into 2 major divisions, the superiorly directed temporo-facial and the inferiorly directed cervicofacial branches → 5 major branches of the facial nerve exist: temporal (ie, frontal), zygomatic, buccal, marginal mandibular, and cervical.
- The facial nerve innervates 14 of the 17 paired muscle groups of the face on their deep side. (The 3 muscles *buccinator*, *levator anguli oris*, *mentalis* may be innervated by buccal and marginal branch of VII or of V3 from superficial surface).
- Frequent connections between the buccal and zygomatic branches exist. The temporal and marginal mandibular branches are at highest risk during surgical procedures and are usually terminal connections without anastomotic connections.

Superficial musculoaponeurotic system

- SMAS is a superficial fascial layer that extends throughout the cervical facial region. In the lower face, invests the facial muscles and:
- Inferiorly, is continuous with the platysma muscle.
- Superiorly, (ends at the level of the zygoma because of attachments of the fascial layers to the zygomatic arch???) continue as temporoparietal fascia. (The temporoparietal fascia extends from the zygomatic arch as an extension of the deep temporal fascia, the frontal branch of the facial nerve crosses the zygomatic arch and courses within the superficial layer of the deep temporal fascia (temporoparietal fascia)) & In the scalp, SMAS is the galea aponeurotica (ensheathes the occipitofrontalis, procerus, and some of the postauricular muscles). In the upper face, the neurovascular structures exit their

bony foramina and penetrate the SMAS to run within its superficial aspects or on its surface.

- The SMAS encloses all of the facial muscles and comprises their only attachment to the overlying dermis, thus transmitting contractions of the facial muscles to the overlying skin.



- The SMAS also helps the surgeon identify the location of the facial nerve:
 - In the lower face, the facial nerve always runs deep to the platysma and SMAS and innervates the muscles on their undersurfaces (except for the *buccinator*, *levator anguli oris*, and *mentalis* muscles???????).
 - In midface, in dessiction toward midline of the face, the nerve can be found running on top of the masseter muscle just below the SMAS.

Temporal (frontal) branches

- The temporal branch of the facial nerve exits the parotid gland and runs within the SMAS over the zygomatic arch into the temple region. The frontal branch enters the undersurface of the frontalis muscle and lies superficial to the deep temporalis fascia.
- To avoid injury to the frontal branch during elevation of facial flaps, the surgeon should elevate either in a subcutaneous plane or deep to the SMAS.

Marginal branches

- The mandibular (or marginal) division lies along the body of the mandible (80%) or within 1-2 cm below (20%).
- This is a critical landmark in head and neck surgery. The marginal branch lies deep to the platysma throughout much of its course. It becomes more superficial approximately 2 cm lateral to the corner of the mouth and ends on the undersurface of the muscles. Injury to the marginal branch results in paralysis of the muscles that depress the corner of the mouth.

Summary of Innervation and Actions of Facial Mimetic Muscles

Branch of CN VII	Location of Lesion	Actions
Posterior auricular	Posterior auricular	Pulls ear backward
	Occipitofrontalis, occipital belly	Moves scalp backward
Temporal	Anterior auricular	Pulls ear forward
	Superior auricular	Raises ear
	Occipitofrontalis, occipital belly	Moves scalp forward
	Corrugator supercillii	Pulls eyebrow medially and downward
	Procerus	Pulls medial eyebrow downward
	Orbicularis oculi	Closes eyelids and contracts skin around eye
Temporal and zygomatic	Zygomaticus major	Elevates corners of mouth

Buccal	Zygomaticus minor	Elevates upper lip
	Levator labii superioris	Elevates upper lip and midportion nasolabial fold
	Levator labii superioris alaeque nasi	Elevates medial nasolabial fold and nasal ala
	Risorius	Aids smile with lateral pull
	Buccinator	Pulls corner of mouth backward and compresses cheek
	Levator anguli oris	Pulls angles of mouth upward and toward midline
	Orbicularis	Closes and compresses lips
	Nasalis, dilator naris	Flares nostrils
	Nasalis, compressor naris	Compresses nostrils
	Depressor anguli oris	Pulls corner of mouth downward
Buccal and marginal mandibular	Depressor labii inferioris	Pulls lower lip downward
Marginal mandibular	Mentalis	Pulls skin of chin upward
Cervical	Platysma	Pulls down corners of mouth

FACIAL NERVE PARALYSIS

- The spectrum of facial motor dysfunction is wide. Several systems have been proposed, but, since the mid 1980s, the **House-Brackmann system** has been widely used. In this scale, grade I is assigned to normal function, and grade VI is complete paralysis.

➤ House-Brackmann Facial Nerve Grading System

Grade	Dysfunction	Characteristics
I	Normal	<i>Normal</i> facial function in all areas
II	Mild	<i>Slight weakness</i> noticeable on close inspection; may have very slight synkinesis
III	Moderate	<i>Obvious, but not disfiguring</i> , difference between 2 sides; noticeable, but not severe, synkinesis, contracture, or hemifacial spasm; complete eye closure with effort
IV	Moderately severe	<i>Obvious weakness or disfiguring asymmetry</i> ; normal symmetry and tone at rest; incomplete eye closure
V	Severe	Only barely perceptible motion; <i>asymmetry at rest</i>
VI	Total paralysis	<i>No movement</i>

- These stages correspond with the pathologic findings of neurapraxia, axonotmesis, neurotmesis, and partial and complete transection of the facial nerve.
 - Grade I → neurapraxia, which is the most likely stage for spontaneous recovery.
 - Grade II, III → axonotmesis, with temporary axonoplasmal flow interruption and subsequent Wallerian anterograde degeneration. Degeneration in axonotmesis is most often incomplete with more or less axons surviving, thus clinically often a partial facial weakness results.
 - Grade IV → Neurotmesis
 - Grade V, VI → partial or complete transection of the facial nerve are either minimal facial musculature movements or complete loss of function.

VASCULAR SUPPLY OF THE FACIAL NERVE

- The cortical motor area of the face is → the *Rolandic branch of the middle cerebral artery*.
- Within the pons, the facial nucleus → *anterior inferior cerebellar artery (AICA)*.
- 3 sources of arterial blood supply to the extramedullary (ie, intrapetrosal) facial nerve:
 - The AICA ← basilar artery, enters the internal auditory canal with the facial nerve → branches into the labyrinthine and cochlear arteries.
 - The *superficial petrosal branch* ← *middle meningeal artery*.
 - The *posterior auricular artery* (at and distal to the stylomastoid foramen).

- Venous drainage parallels the arterial blood supply.

Monitors, Facial Nerve

Electromyography depends on noting the difference in electrical potential that occurs within the muscle associated with a depolarizing current. As the electrical potential moves past the first of the 2 paired electrodes, that electrode becomes negative with respect to the second. As the wave reaches the second electrode, a deflection in the opposite direction occurs. The electromyographic electrical response is biphasic, ie, it has both positive and negative components.

Prass has distinguished between 2 types of potential responses.

Repetitive responses

as a result of repetitive depolarizations that follow the cessation of surgical manipulation. (indicate ↑ irritability as a result of injury).

Nonrepetitive responses

Nonrepetitive responses are produced by direct mechanical or electrical stimulation of the facial nerve and occur in close temporal association with the stimulus. (are much more important in defining the boundaries of the nerve) → allow the surgeon to locate the edge of the nerve and to map its anatomic contour.

Equipment setup

- Pulse duration
 - The default setting for most physiologic monitors is 100 microseconds. Selesnick has shown that 50 microseconds provides a more sensitive stimulus.
 - Some surgeons use a pulse duration of 200 microseconds.
- Monopolar versus bipolar stimulation
 - Bipolar stimulation is more precise because less opportunity exists for shunting of current away from the site of stimulation. However, it is much more prone to false-negative results because no stimulation occurs unless the nerve lies directly in the current's path.
 - Monopolar stimulation is more common than bipolar, but both techniques are used.
- Constant current versus constant voltage
 - Ohm law indicates that if constant voltage stimulation is used, voltage increases to compensate for current shunting.
 - Nonetheless, constant current systems are used more commonly.
- Stimulus intensity
 - The appropriate stimulus intensity may vary considerably. Unless injured, the unsheathed facial nerve in the subarachnoid space reliably responds to a stimulus intensity as low as 0.05 mA, although the sheathed facial nerve in the mastoid segment often does not respond to stimulus intensities less than 0.15 mA. The operating surgeon must be familiar with this variability.
- Threshold
 - The threshold at which the monitor produces an audible response can be set for most facial nerve monitors and is generally set at 100 microvolts.

CLINICAL APPLICATION - ELECTRODE PLACEMENT

Electrode placement

Paired electrodes are placed in 2 facial muscle groups. The portion of the electrode not within the muscle itself should be insulated. The orbicularis oris and the orbicularis oculi are usually selected.

Once all the electrodes are properly positioned and placed, tapping gently on the face to determine whether a faint response can be heard from the facial nerve monitor is useful. A response indicates that the system is functioning properly.

Monitoring

The amount of electromyographic feedback that can be expected during a given operative procedure depends on the following variables:

- The adequacy of intramuscular electrode placement
- The number of independent EMG channels
- The effectiveness of nerve stimulation
- The level of nerve irritability
- The conduction status of the nerve distal to the point of stimulation

The facial nerve monitor can then be used during the course of dissection for the following purposes:

- To identify the facial nerve
- To map its contour
- To identify potential injurious stimuli
- To obtain prognostic information about postoperative facial nerve function

Various authors have indicated that a compound action potential at the end of the procedure that exceeds 500-800 microvolts indicates a high likelihood of a House-Brackman (HB) I or II facial nerve outcome. The incidence of permanent facial weakness increases as the amplitude of the compound action potential drops below 500 microvolts.

Facial Nerve Paralysis

Pathophysiology:

- Facial nerve injury can be complete or partial.
- Loss of motor function can be observed immediately after facial nerve injury. Depending on the affected trunk and localization (proximal or distal), various patterns of motor function loss can be seen and used for primary diagnosis of the lesion site.
- Significant muscle fiber decay has been demonstrated when denervation has been present for more than 3 years.
 - Early changes at cellular level (1 week after denervation) include chromatin changes and increased mitochondria number, DNA, and satellite cells, thus reflecting the plastic state of denervated muscle.
- In addition to clinical and Histopathologic Findings, parasympathetic functions such as salivation, lacrimation, and taste sensation also may be impaired.

Causes of Facial Nerve Palsy (1900-1990)

Birth	Molding
	Forceps delivery
	Dystrophia myotonica
Trauma	Möbius syndrome (facial diplegia associated with other cranial nerve deficits)
	Basal skull fractures
	Facial injuries
	Penetrating injury to middle ear
	Altitude paralysis (barotrauma)
Neurologic	Scuba diving (barotrauma)
	Lightning
	Opercular syndrome (cortical lesion in facial motor area)
	Millard-Gubler syndrome (abducens palsy with contralateral hemiplegia caused by lesion in base of pons involving corticospinal tract)
Infection	External otitis
	Otitis media
	Mastoiditis
	Chickenpox
	Herpes zoster cephalicus (Ramsay Hunt syndrome)
	Encephalitis
	Poliomyelitis (type 1)
	Mumps
	Mononucleosis
	Leprosy
	Influenza
	Coxsackievirus
	Malaria
	Syphilis
	Scleroma
	Tuberculosis
	Botulism
	Acute hemorrhagic conjunctivitis (enterovirus 70)
	Gnathostomiasis
	Mucormycosis
Metabolic	Lyme disease
	Cat scratch
	AIDS
	Diabetes mellitus
	Hyperthyroidism
	Pregnancy
	Hypertension
	Acute porphyria

Neoplastic	Vitamin A deficiency
	Benign lesions of parotid
	Cholesteatoma
	Seventh nerve tumor
	Glomus jugulare tumor
	Leukemia
	Meningioma
	Hemangioblastoma
	Sarcoma
	Carcinoma (invading or metastatic)
	Anomalous sigmoid sinus
	Carotid artery aneurysm
	Hemangioma of tympanum
	Hydradenoma (external canal)
	Facial nerve tumor (cylindroma)
	Schwannoma
	Teratoma
	Hand-Schüller-Christian disease
	Fibrous dysplasia
	Neurofibromatosis II
Toxic	Thalidomide (Miehlke syndrome, cranial nerves VI and VII with congenital malformed external ears and deafness)
	Ethylene glycol
	Alcoholism
	Arsenic intoxication
	Tetanus
	Diphtheria
Iatrogenic	Carbon monoxide
	Mandibular block anesthesia
	Antitetanus serum
	Vaccine treatment for rabies
	Postimmunization
	Parotid surgery
	Mastoid surgery
	Post-tonsillectomy and adenoidectomy
	Iontophoresis (local anesthesia)
	Embolization
Idiopathic	Dental
	Familial Bell palsy
	Melkersson-Rosenthal syndrome (recurrent alternating facial palsy, furrowed tongue, faciolabial edema)
	Hereditary hypertrophic neuropathy (Charcot-Marie-Tooth disease, Dejerine-Sottas disease)
	Autoimmune syndrome
	Amyloidosis
	Temporal arteritis
	Thrombotic thrombocytopenic purpura
	Periarteritis nodosa
	Landry-Guillain-Barré syndrome (ascending paralysis)
	Multiple sclerosis
	Myasthenia gravis
	Sarcoidosis (Heerfordt syndrome, uveoparotid fever)
	Osteopetrosis



CLINICAL

Clinical diagnosis is based on 3 steps:

1. identification of the affected site
2. underlying etiology (trauma, infectious, neoplastic)
3. clinical staging

History

onset of symptoms, an evaluation of the quality of associated symptoms, and PH of infections and systemic diseases.

Physical:

- A thorough head and neck examination is paramount, with occasional use of tests for salivation, tearing, and taste; these are the first steps in determining the site of injury.
- Physical examination findings reveal affected facial musculature movement.
- Tests for facial innervation include the following:
 - Forehead wrinkling (frontalis muscle)
 - Eye closure (orbicularis oculi muscle)
 - Wide smile
 - Whistling
 - Blowing (eg, buccinator muscle, orbicularis oris muscle, zygomatic muscle)
- Evaluate general muscle status (latissimus muscle, rectus abdominis muscle) for eventual reconstruction.
- Grading by House-Brackmann scale.
Table 1. House-Brackmann Classification of Facial Function*
- A common entity of facial nerve paralysis is Bell palsy, a form that is unilateral and considered of idiopathic etiology. Incidence 20 cases / 100,000
 - Bell palsy normally has a sudden onset often preceded by facial dysesthesia, epiphora, pain, hyperacusis, dysgeusia, and decreased function of the lacrimal gland (May, 1991).
- Ramsay Hunt described a syndromic occurrence of facial paralysis, herpetiform vesicular eruptions, and vestibulocochlear dysfunction:
 - have a greater risk of hearing loss than patients with Bell palsy, and the course of disease is more painful. Moreover, a lower recovery rate is observed in these patients.
 - Medical treatment is equivalent to Bell palsy; most often a combination of steroids and antiviral agents is used.

WORKUP

Imaging Studies:

- CT and MRI

Other Tests:

- Electrophysiology can be useful to determine the extent of nerve disruption: Most frequently, the minimal and maximal stimulation test (MST) and electroneuronography (ENog) are used. (with percutaneous stimulation of the facial nerve):
 - ENog studies are required to determine timing and necessity of surgical intervention (decompression or microneurorrhaphy).
 - ENog records a compound action potential (CAP) as well as latency after nerve stimulation. Degeneration of 90% or more has been shown to predict poor prognosis without surgical intervention.
- For suspected intracranial or infratemporal injury, always perform a Schirmer test of tearing to assess lacrimal gland function.
- Testing with different aromatic agents is needed to determine the integrity of afferent impulses from the anterior $\frac{2}{3}$ of the tongue.

TREATMENT

Medical Care:

- Management of synkinesis and hyperkinesis can include Botox® injection. procedures but must be repeated approximately every 3 months. (5-10 units are injected initially to

control eyebrow spasm and an additional 10-20 units are injected into the zygomaticus muscle and then repeated with an adapted dose as needed).

Surgical Care:

- The patient's facial appearance mainly is impaired by loss of muscle tone on the affected side but it is also influenced by severe contraction on the opposite healthy side.
- **Goals of reconstruction of the paralyzed face include the following:**
 1. Facial symmetry at rest
 2. Adequate facial function, including *oral* competence and eye closure
 - Voluntary facial movement
 - Spontaneous facial expression
 3. Absence of synkinesis or mass movement
- **Options include:**
 - a) Dynamic facial reanimation procedures:
 1. direct coaptation
 2. interposition nerve grafting (Donor nerves: the ansa hypoglossi, sural nerve, and medial cutaneous antebrachii nerve)
 3. cross-face nerve grafting
 4. microneurovascular free tissue transfer.
 - b) Static procedures: After these, balancing and adjustment procedures are performed to give the face the final desired symmetry.
 1. operations on the depressor anguli oris muscle group, lip wedge resection.
 2. enhancement of the nasolabial fold.
 3. static eye procedures such as upper eye lifting, lateral canthopexy, static sling placement.
 4. partial cervicofacial rhytidectomy.
- Follow up is important, e.g. in cross-facial nerve grafting, use the sign of Tinel (paraesthesia when tapping on the regenerated end of the graft) for monitoring the nerve regeneration along the graft & care in microneurovascular tissue transfer, the method described by Fasching and Van Beek, who placed electrical monitoring devices into the grafted muscle.
- **Timing:**
 - Acute facial nerve palsy (< 1 year) must be subclassified as acute nerve injury secondary to direct trauma or injury due to facial surgery (inadvertent transection or sacrifice for oncologic reasons)
 - immediate reconstruction: direct anastomosis (microneurorrhaphy) or interposition nerve grafting.
 - Direct anastomosis principles:
 - microsurgical technique
 - without undue tension to minimize scar formation (↓ axonal regeneration).
 - Fascicle sutures are theoretically possible, but no evidence supports the superiority of this technique compared to the epineural suture???
 - However, synkinesis, facial spasm, and mass movement remain frequent complications in rehabilitation of the facial nerve.
 - Interposition nerve graft: If tension-free coaptation cannot be performed.
 - The great auricular nerve often can be used. This nerve is harvested using an incision made in an imaginary line drawn from the mandibular angle posterior to the mastoid tip. (provides sensation to the postauricular and the posterolateral cervical area).

- the sural nerve. (supplies sensation to the lateral aspect of the calf) usually is harvested by several small incisions cranial from approximately 1 cm posterior to the lateral malleolus. Its major advantage vis-à-vis the great auricular nerve is its length, (upto 35cm).
- the ansa hypoglossi is used when a combination of parotidectomy and neck dissection is performed. In this case, no new skin incision is needed, and the oncologically sacrificed distance of the facial nerve can be adapted exactly to the donor nerve harvest length (ansa hypoglossi). Also, the nerve transfer of partial ansa hypoglossi nerve to the facial nerve can be performed with good results. A partial nerve transfer can reduce donor nerve complications (difficulties with speech and mastication).

- Regenerative impulses yield an axonal length gain of 1-1.5 mm/day (1inch/month)
- Muscle tone and movement is regained approximately 6-9 months after grafting.
- Muscle atrophy occur if not reinnervate in 18 month

- Chronic facial paralysis (> 1year)

Two types of procedures are used, *dynamic reanimative and static procedures*.

- Carefully evaluate the patient's remaining potential for spontaneous recovery by electromyography (EMG), MST, and ENog.
- upper 1/3 of the face, a frequent problem is lack of eye closure and ectropic lower eyelid;
 - placement of upper-lid gold weight is performed as a static procedure.
 - For lower-lid ectropion, wedge excision of the lateral lower eyelid and lateral canthopexy as static procedures.
 - Cosmetic impairment as found in ptosis of the eyebrow can be corrected with partial forehead and browlifts.
- The mid 1/3 of the face is the most challenging region in facial reanimation.
 - staple static procedures such as slings (either temporalis sling or Gore-Tex sling) and cervicofacial rhytidectomy
 - more complex reconstruction such as tissue transfers or cross-facial reinnervation procedures.
 - Depend on the patient's personal needs and, the patient's general health.
 - eg, diabetes, old age, multimorbid state)→ static procedures.
 - the patient is in general good health and seeks dynamic rehabilitation. Two choices are possible with these patients. First of all, obtain an EMG percutaneous test.

1. If EMG testing reveal **polyphasic motor unit potentials (MUP)**, [i.e. regenerative processes of the facial nerve]→ Direct coaptation of the proximal and distal stumps or interposition nerve grafting should not be performed at this time → close follow-up study of nerve regeneration with clinical and technical methods (physical examination, Tinel sign, ENog, MST) is the therapeutic strategy of choice.
2. If EMG testing reveal **fibrillation potentials**. [i.e. the permanent denervation of the facial nerve] → coaptation of the distal and proximal stump is indicated → If the stumps cannot be approached easily intraoperatively, farther proximal preparation can be performed to gain more length → Cable grafting (interposition nerve grafting using either the greater auricular nerve or sural nerve).

3. **Silence** on the EMG [i.e. long-term denervation]. → coapting the stumps is not successful → innervated free tissue transfer or muscle transposition (temporalis, masseter, gracilis muscle) is indicated. The performance of microvascular tissue transfer has two major advantages in this scenario, the possibility of voluntary facial musculature movement and volume gain in case of loss of muscle volume after surgery.
- **The crossover facial reinnervation technique**
 - when only the *distal stump of the facial nerve is viable*.
 - The most frequently used donor nerve is the hypoglossal nerve. However, the patient may experience swallowing and speech difficulties due to the lack of hypoglossal nerve motor input → can be minimized by transposition of only approximately one half of the hypoglossal nerve. Also, jump grafting (interposition) with the use of the hypoglossal nerve to coapt the facial nerve by interposition of a nerve graft.
- **VII-VII transfer**
 - A well-known technique is the coaptation of the contralateral buccal branch to the ipsilateral facial nerve. 2stages:
 - Primarily, a sural nerve graft is coapted to the contralateral buccal branch, then tunneled through the upper lip and usually left in the subcutaneous tissue. This is performed to allow axonal regeneration on the sural nerve. Closely monitor for axonal regeneration (Tinel sign)
 - However, the results of this technique are not favorable; additionally, the patient is at risk of losing innervation of the contralateral mid face.
- **Muscle transposition**
 - The temporalis muscle often is used for this transposition technique.
 - Technically, elevate a temporo-fascial flap using a hemi-coronal skin incision. Incise a strip of approximately 2 cm and rotate it distally through a preformed subcutaneous tunnel down to the mesiolabial fold. Here, connect the flap to the upper lateral part of the orbicularis oris muscle. Overcorrection is of the utmost importance because of possible postoperative tissue laxity.
 - Another muscle for possible transposition is the masseter muscle, which is not used frequently due to the concomitant complications in mastication and speech.
- **Microneurovascular free tissue transfer**
 - frequently is used after tumor surgery of the face:
 - restoring voluntary facial movement
 - reconstructing and/or filling the soft tissue defect.
 - Advantages over muscle transposition:
 - excellent results in postoperative movement of the face, mastication, and speech.
 - possibility of reconstruction of spontaneous facial musculature movement by coaptation to the proximal facial nerve stump.
 - restore large volume defects that could not be reconstructed easily with the comparable "low-volume" temporalis muscle transposition.
 - Both the **gracilis muscle** free flap and the **latissimus dorsi muscle** flap are frequently.

- The **rectus abdominis muscle free flap** can be harvested in a standard fashion as needed either $\pm$ a skin paddle. Muscle transfer and preparation depends on the needed tissue volume and length.
- Another excellent muscle for free microvascular transfer is the **pectoralis minor muscle**. (originates from the 3rd → 5th ribs, has the major advantage in that it has a strong tendinous insertion, which makes it ideal for a "pull-up" muscle for the restoration of a smile. Moreover, the pectoralis minor muscle has dual nerve supply (both the lateral and medial pectoral nerves), making single-stage smile and eye closure restoration possible by splitting the flap.
- After dissection of muscle paddles, the flap is transferred up to the face and fixed to the zygomatic arch, lateral corner of the mouth, and second paddle from the lateral canthus to the medial, thus reanimating both the middle part of the face (smile reconstruction) and the upper part (eye closure).
- Overcorrection is essential in both the gracilis muscle free flap and the rectus abdominis muscle free flap.
- Lower lip paralysis can be corrected with a **static sling**.
 - usually is harvested from the anterior thigh fascia and adapted to the lateral orbicularis oris muscle and the zygomatic arch as previously described.
 - Gore-Tex slings are described in the literature as well.
- Moreover, "common" aesthetic procedures such as partial cervicofacial rhytidectomies, cheiloplasty, and brow lift can be offered to the patient as ancillary or "touch-up" procedures for the restoration of the paralyzed face.
- Downward deviation of the lateral lip aspect often requires lateral lip wedge resection. This procedure often must be combined with static sling procedures or deep plane face lift to gain acceptable results in facial symmetry.

FOLLOW-UP

Follow up and Consultations:

Physical rehabilitation: The physical therapist should teach the patient how to innervate the facial muscle efficiently after nerve transfer and grafting. Also, the patient should be encouraged to exercise the facial musculature to gain maximum strength of muscle pull. Nerve stimulation can be used postoperatively; however, electrical stimulation does not constantly demonstrate evident improvements.

Further Inpatient Care:

- In microvascular transfers, follow-up care involves close monitoring in the hospital with Doppler signals and thorough examination of the flap, Capillary refill and the pinprick test, thermocouple devices.

Further Outpatient Care:

- For interposition or cross-facial nerve grafts, the Tinel sign can be observed along the course of the regenerating nerve, approximately 1 mm/d. Nerve conduction studies also can be used as adjunct studies.
- For static procedures, clinical examination is the basis of follow-up care. Closely monitor overcorrection, since both gravity and skin laxity should equalize facial hemispheres by approximately 6 months postoperatively.

Complications:

- free muscle transfer for facial paralysis: Complications observed in 11%
 - Arterial thrombosis in 5%

- Venous thrombosis in 3%
- Complete arterial and venous occlusion (1%)
- Hematoma (3%)
- Failure of muscle transplantation (4%)
- Muscle necrosis (1%)
- No signs of reinnervation; improvement after reexploration

Congenital Facial Paralysis

Frequency: 0.23-1.8% of live births. Of these births, 78-90% are associated with birth trauma. Congenital unilateral lower lip palsy (also referred to as velocardiofacial [VCF] syndrome in some references) is the most common of the developmental lesions, occurring in 1 out of 120-160 live births.

Etiology: Congenital facial paralysis (traumatic or developmental causes)

- traumatic causes are related primarily to a difficult labor.
- developmental facial nerve paralysis are numerous and include mononeural agenesis, congenital paralysis, and congenital unilateral lower lip paralysis (CULLP). When CULLP occurs with cardiac anomalies, the condition is referred to as VCFL syndrome in some references.

Deficits associated with facial nerve paralysis

- Möbius syndrome:
- Hemifacial microsomia:
- Poland syndrome:
- Goldenhar syndrome: This syndrome is a nonhereditary congenital variant of hemifacial microsomia. This condition includes additional findings of vertebral anomalies and epibulbar dermoids.
- DiGeorge syndrome
- Albers-Schönberg disease: Osteopetrosis, a rare cause of paralysis at birth, may manifest later in childhood.
- Trisomy 18, trisomy 13
- CHARGE syndrome: This acronym stands for colobomata, heart disease, atresia of choanae, retarded growth, genital hypoplasia, and ear anomalies. Multiple cranial nerves may be involved (60% → VIII, 43% → VII, and 30% → IX and X).
- Muscular dystrophy.

Systemic or infectious conditions associated with facial nerve paralysis

- Melkersson-Rosenthal: This condition involves recurring attacks of unilateral or bilateral facial paralysis, swelling of the lips, and furrowing of the tongue. Angiotensin-converting enzyme is elevated during attacks.
- Poliomyelitis
- Infectious mononucleosis
- Varicella
- Acute otitis media
- Mastoiditis
- Meningitis
- Bell palsy

Teratogens associated with facial nerve paralysis

- Thalidomide embryopathy: This sedative is administered at 28-42 weeks' gestation and is associated with phocomelia, arrested development of the ear, and paralysis of the facial and abducens nerves.
- Misoprostol: This synthetic prostaglandin E1 analogue is used to prevent and treat gastric ulcers and gastrointestinal lesions induced by nonsteroidal anti-inflammatory drugs (NSAIDs) → Möbius syndrome & Neural tube defects

Medical therapy:

- 1- eye care (artificial tears, ointment)
- 2- traumatic congenital facial paralysis→ observation and corticosteroids. (90% recover spontaneously).

Surgical therapy:

- no procedures are available that can enable an infant to develop normal function of the facial nerve when the palsy is developmental in origin. Surgical exploration in the newborn with facial paralysis is controversial.

Neurorrhaphy

The best situation for repair of the facial nerve is when primary reanastomosis is possible between the transected ends;

Cable grafts

In situations in which the nerve has been crushed and neurorrhaphy cannot repair it, a cable graft may be indicated. The most common donor nerves are the greater auricular and sural nerves.

Cross-face grafts

on the contralateral face. This technique may be combined with microvascular muscle grafts.

Nerve transposition

This procedure is indicated when no known proximal facial nerve is available based upon MRI evaluation, physical examination, and topodiagnostic studies. The hypoglossal nerve provides the best crossover graft with minimal resultant lingual atrophy. Facial nerve-hypoglossal nerve grafts are not indicated in developmental palsy because of the impairment of the distal peripheral nerve and neuromuscular junction. An ideal outcome of this technique is good symmetry at rest, some voluntary movement with synkinesis, and mass movement; however, no emotional facial expression is expected.

Muscle transfer**Static sling****Eye protection**

When eye protection is inadequate and corneal abrasions result, tarsorrhaphy, gold weights, and palpebral springs should be considered. Gold weights are likely the best option because they are simple to insert and easily removed. This procedure is rarely performed in the newborn because parents are often very capable of protecting the infant's eyes.

Treatment for (congenital lower lip paralysis (CULLP))

Several options are specific to CULLP. Most parents do not notice any defect except when the child is crying; therefore, surgical intervention in the isolated CULLP deformity is rarely indicated.

Surgical procedures to weaken the nonaffected side with selective marginal mandibular neurectomy or botox injections provide symmetry at rest. Other plastic reconstructive options include wedge resection and fascia lata sling or cheiloplasty, plication or transposition of the orbicularis oris muscle, and digastric muscle transfer.

**FUTURE AND CONTROVERSIES**

Much controversy exists regarding the timing of facial reanimation and the need for surgical exploration in children with congenital facial paralysis. Issues regarding the timing of reanimation are complex. Some health professionals advocate initial surgery during preschool to prevent the psychosocial aspects associated with a physical abnormality.

However, waiting until adolescence when facial growth is mature and the child is able to understand the risks and benefits of surgery and participate in the decision making process also has merit.

Summary and Notes

The procedures for total unilateral facial paralysis are as follows:

- ▶ Direct facial nerve anastomosis
- ▶ Interpositional grafts
- ▶ Anastomosis to other motor nerves
- ▶ Dynamic musculofascial transpositions
- Static musculofascial transpositions
- Facial plastic procedures

The first 4 are dynamic procedures that restore some voluntary movement and, thus, are more desirable. The latter procedures are reserved for patients in whom the motor end plates are not viable. Combinations of the above procedures may be appropriate depending on the circumstances.

Principles of Nerve Repair

- ☑ Early identification and repair of nerve injuries
- ☑ Evaluation of nerve condition
- ☑ Matching of endoneurial surfaces
- ☑ Epineural versus perineural suturing
- ☑ Tension-free anastomosis and nerve grafts

Location of Injury

- Intracranial - Agenesis or congenital abnormality, CNS degeneration, trauma, tumor, vascular abnormality
- Intratemporal - Cholesteatoma, iatrogenic, infectious, trauma, tumor
- Extratemporal - Iatrogenic, trauma, tumor

NERVE SUBSTITUTION

Donor Nerves for Facial Nerve Grafting → Great auricular nerve and sural nerve

Donor nerves available for substitution → spinal accessory, phrenic, trigeminal, and hypoglossal nerves. Another substitution technique is cross-facial nerve grafting.

Selection of Patients

- ☑ Irreversible facial nerve injury
- ☑ Intact mimetic function
- ☑ Intact motor end-plate function
- ☑ Intact proximal donor nerve
- ☑ Intact distal facial nerve

Techniques

- ☑ Direct hypoglossal-to-facial graft
- ☑ Partial hypoglossal-to-facial jump graft
- ☑ Cross-facial graft

MUSCLE TRANSPOSITION

Muscles Available for Regional Transposition

- ☑ Temporalis

☑ Masseter

☑ Digastric

Indications:

- An intact facial neuromuscular system is absent, such as in congenital facial paralysis (Möbius syndrome).
- Longstanding facial nerve interruption (at least 3 y) results in loss of motor end plates.
- Other cranial nerves are sacrificed; therefore, a crossover technique cannot be tolerated.

Free-Muscle Transfer

☑ Principles of Free-Muscle Transfer

☑ Nerve graft and free muscle flap

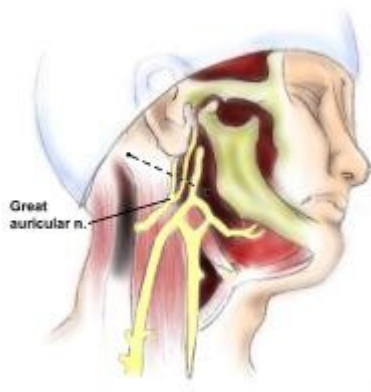
☑ Cross-facial nerve graft

☑ Muscles suitable for transfer

Terminal branches of the facial nerve, demonstrating its variability
B – buccal M – mandibular T – temporal Z – zygomatic



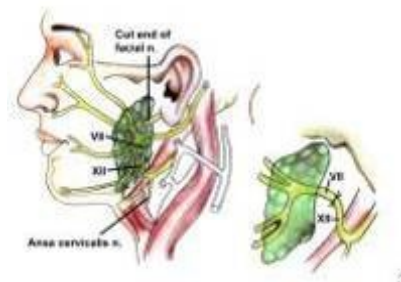
Greater auricular nerve located superficial to the sternocleidomastoid, perpendicular to a line drawn between the mastoid and the angle of the mandible



Sural nerve located just lateral to the saphenous vein and medial and posterior to the lateral malleolus of the ankle

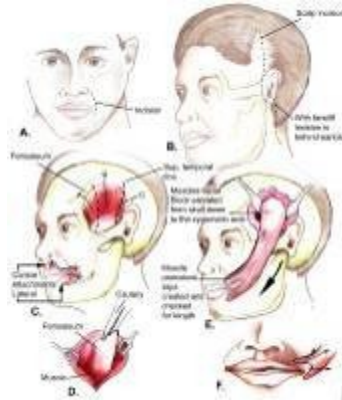


Nerve crossover using the proximal trunk of the hypoglossal nerve to the distal trunk of the facial nerve



The temporalis muscle transfer

- Nasolabial incision
- Scalp incision incorporated with facelift incision behind the ear
- Superimposed incisions over the temporalis muscle indicating a harvest of the middle portion of the temporalis muscle
- Incision of the temporalis muscle superiorly, separating the periosteum from the skull base
- Transfer of the temporalis muscle in a subcutaneous plane, but superficial to the muscular aponeurotic system.
- Insertion of the temporalis muscle into the orbicularis oris muscle with an overcorrected position

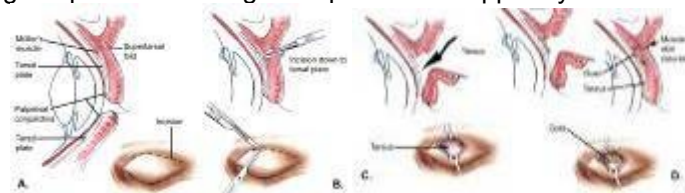


- Intraoral approach harvests the masseter muscle for transfer. Incision is made along the gingival sulcus.
- One muscle is exposed; curved scissors are used to transect the muscle in the midportion.
- Two slips of muscle are attached to the dermal layers of the skin for overcorrection of the smile.



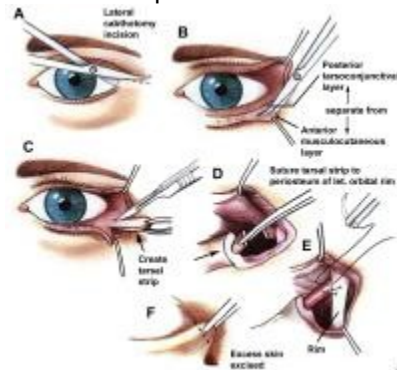
Gold implant technique for upper eyelid closure

- Incision is made several centimeters above the upper eyelid.
- With a sharp instrument, the tarsal plate is identified.
- The gold implant is sutured in place, straddling the tarsal plate and slightly posterior to it.
- Lateral view is showing the position of the gold implant in the upper eyelid.



Lateral tarsal strip procedure for ectropion of the lower lid

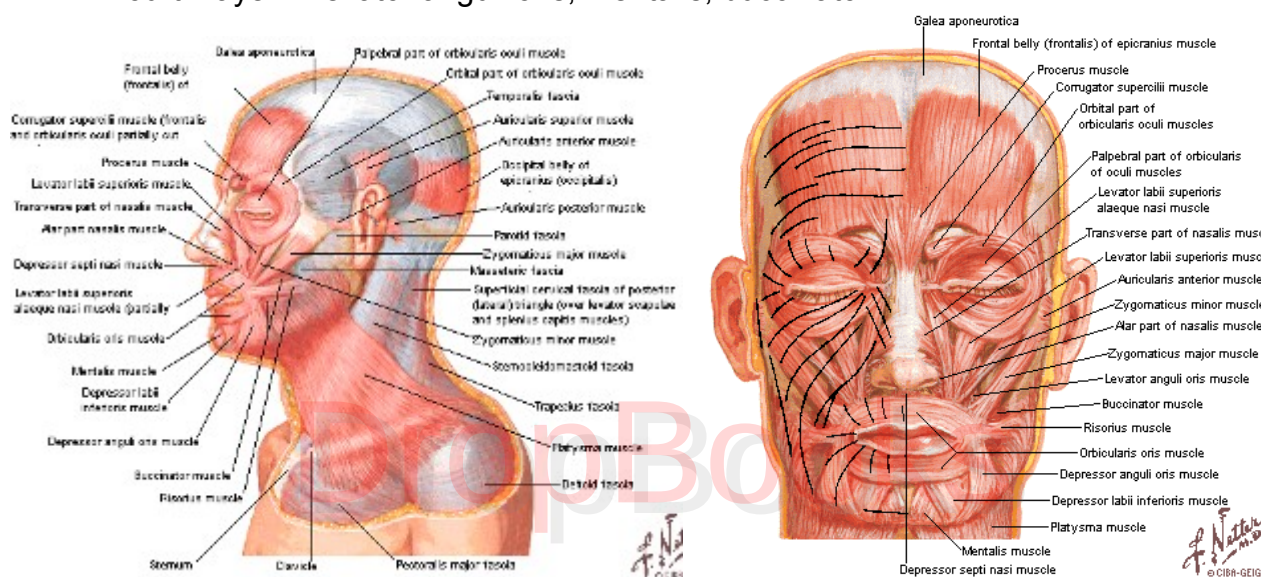
- A lateral canthotomy incision is shown.
- Division of the lateral aspect of the lower lid into an anterior musclocutaneous layer and posterior tarsal conjunctival layer is shown.
- Tarsal strip is grasped with skin hook.
- Tarsal strip is positioned inside the lateral rim of the orbit, which has been exposed.
- Tarsal strip is sutured to periosteum inside of lateral orbital rim.
- Excess skin is excised and wound closed



RELEVANT ANATOMY AND CONTRAINDICATIONS

Relevant Anatomy: The mimetic muscles of the face are arranged in 4 layers. The muscles in the first 3 layers are innervated on their deep surfaces while the muscles of the fourth layer are innervated at their superficial surface. The main branches of the facial nerve are arranged consecutively deeper. The temporal branch is the most superficial while the zygomatic, buccal, and marginal branches lie in deeper layers. The muscles that comprise the 4 layers are as follows:

- First layer - Depressor anguli oris, superficial portion of zygomaticus minor, orbicularis oculi
- Second layer - Platysma, risorius, zygomaticus major, deep portion of zygomaticus minor, levator labii superioris alaeque nasi
- Third layer - Levator labii superioris, orbicularis oris
- Fourth layer - Levator anguli oris, mentalis, buccinator



Contraindications: to dynamic facial reanimation exist, as follows:

- (1) absence of facial nerve branches or mimetic muscles of the face,
- (2) swallowing dysfunction,
- (3) denervated or excised temporalis and/or masseter muscles,
- (4) compromised facial vasculature.

Contraindications for dynamic reanimation techniques

- Ablation of distal facial nerve branch - Cable graft or nerve crossover CN X/VII
 - Absence of mimetic muscle (or atrophy) - Cable graft or nerve crossover CN X/VII
 - Damage to VII at brain stem - Cable graft or nerve crossover CN X/VII
 - Swallow dysfunction - Nerve crossover CN IX/VII
 - Absent or denervated temporalis - Muscle transposition
 - Absent or denervated masseter - Muscle transposition
 - Vascular compromise - Microvascular free flap
- Static reanimation can be accomplished with fascia or alloplastic material.
 - Static reanimation allows for repositioning of the tissues that are affected by the pull of gravity. Resuspension by sling with multidirectional traction helps compensate for the complex contractions of the facial mimetic muscles

- Static reanimation may be an option for the debilitated patient who cannot withstand a longer operative procedure such as microvascular free flap or neural grafting techniques.

Indications for static procedures

- Management of the paralyzed eyelid
- Alternatives to dynamic reconstruction in select patients
- Complementary procedures to enhance symmetry

Static procedures:

A- Eyelid and Lacrimal Function

■Orbicularis oculi

■Upper lid

■Lower lid

■Lacrimal gland

Management Strategy

- Supportive therapy
- Tarsorrhaphy
- Gold weight lid loading
- Lower lid procedures

B- LOWER FACE

- Oral commissure and lip suspension
- Nasal lateralization

Disruption of the buccal branches of the facial nerve results in paralysis of the nasalis muscle with collapse of the nostril. Patients may complain of unilateral nasal airway obstruction. Septorhinoplasty may address additional nasal deformities and provide relief of obstruction, but occasionally a static lateralization procedure may be warranted. This procedure may be performed through an external approach, in which an incision is made in the melolabial sulcus and carried around the lateral nasal ala, or through an intraoral approach via an incision made in the upper buccal sulcus. In either approach, suture a strip of fascia lata to the deep tissue of the lateral alar base, pull it laterally, and fix it to the lateral ascending buttress of the maxilla with nonabsorbable suture or by a titanium miniplate.

- Cheiloplasty

C- ADJUNCTIVE PROCEDURES

Browlift, blepharoplasty, and rhytidectomy procedures